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THE MAXIMUM PERMISSABLE EXPOSURE TO RADIATION

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Introduction

Ever since the harmful effects of high frequency radiation on human beings were first noticed, safety rules have been laid down to prevent technicians and patients from being exposed to harmful doses. With the passing of time there has been a tendency to define a smaller and smaller maximum permissable exposure rate, popularly called 'tolerance dose'. This is generally defined as the maximum intensity of radiation to which it is considered 'safe' to be exposed for an indefinite period. However a smaller maximum allowable exposure means correspondingly greater protective measures, and more sensitive measuring devices—a considerable problem for those who wish to follow the safety code, but have not infinite financial resources. Proposals to reduce the allowable exposure still further [1] are therefore viewed critically by some. At a recent round table discussion between radiologists and physicists [2], the latter were asked on what evidence the maximum allowable exposure was chosen, and what would be the effects of surpassing it by modest amounts. Some radiologists expressed the opinion that the present safety code was quite empirical, and probably too conservative 'by a factor of twenty, anyway'. They expressed complete willingness to expose themselves and their technicians to greater than maximum allowable exposure, and observed that this was common practice in many places.

It is true that at the beginning, the maximum allowable exposure was defined more by educated guesswork than by precisely measured evidence. However there has been accumulated of late a great deal of data which would enable the allowable exposure to be established less empirically. This data is scattered in the literature, but a review of it indicates that the educated guessers were not overly conservative.

The Exposure Units

One reason for some misinterpretations of safety code rules is a lack of complete appreciation of the various units used, and the impression that "for all practical purposes the units are identical." Actually, the various units exist for definite reasons, and in many cases are far from identical.

The Roentgen (*r*) is the unit most commonly used to measure radiation dosage. It is a unit of *ionisation intensity in AIR* and is the amount of X or gamma and associated secondary radiation producing one electrostatic unit of charge in one cubic centimeter of dry air under standard conditions, by ionising the air molecules.[3] Taking 32.5 electron volts as the best average value of the energy required to produce an ion pair in air, we find that 1 r corresponds to an expenditure by the radiation of about 83 ergs per gram ($^3/_4$ liter) of air.

It must be remembered that the roentgen is not a unit of radiation intensity. For example, a beam intensity of 100 ergs/cm^2 of 20,000 Volt X-rays is required to produce 1 r, while for 200,000 volt rays, the beam intensity for producing 1 r must be 3,000 ergs per cm^2. Still less is the roentgen a unit of biological effect. Neither does it apply to ionisation intensity produced by other agents than X or gamma rays.

The Rep. If animal matter is placed in a region of air where X or gamma radiation was producing 1 roentgen per unit time interval, the radiation will now ionise the animal matter instead of the air. Due to the differences in absorption coefficients, the radiation will now expend, say, 95 ergs per gram of matter per unit time, if it is muscle. It will expend about 89 ergs per gram if the matter is fat, and about 160 if it is bone. It was expending 83 in the air. Therefore the dosage rate in roentgen units for air does not indicate the energy expended in animal matter unless we know the relative specific ionisations in the various parts of the substance. The matter is further complicated by the variation of these figures with radiation energy. Thus the 160 ergs per gram of bone corresponds to radiation of about 800 Kev energy. A lower energy beam—say 12 Kev—expends about 900 ergs per gram in bone, if the ionisation is to correspond to 1 r, or 83 ergs in air. The coefficients, which are thus a function of tissue type and beam energy, are known for some cases, [4,5] and the calculations are simple.

As a numerical example, if a physician decides that two tumors—one in bone, and one on the skin—may each be benefited by the administration of identical doses (i.e. equal absorptions of

energy), and proposes to make the treatment with a 100,000 volt X-ray machine, he will calculate that for the skin cancer his ionisation chamber should indicate, say, 1 r per second, while for the *same* dose to bone, it should indicate only about 0.3 r per second. He would correct these figures, of course, for back-scatter and absorption.

However this differential absorption makes comparisons inconvenient. To enable direct comparisons of energy absorption in different tissues to be made, a unit called the roentgen-equivalent-physical (rep) has come into use. A rep is the amount of primary and associated secondary radiation of *any kind* (not only X and gamma) which expends 83 ergs per cubic centimeter of tissue.[6] One c.c. of tissue is closely equal to 1 gram. Thus, as previously mentioned, 1 roentgen of X radiation expended in muscle would be about 1.1 rep. As another example, a dose of 100 rep in bone, would be supplied by only 9.2 r of 12 Kev X-radiation. It is thus dangerous to assume the numerical equivalence of the roentgen and the rep. In practice, the former is more easily measured, and the latter must be calculated.

For X and gamma radiation, this calculation is made by measuring the corresponding air dose in roentgens, and multiplying by the relative specific ionisations in the particular tissues involved for the radiation energy used. Corrections are made for back-scatter and absorption [7]. For corpuscular radiation the dose in rep only may be calculated from the number of particles absorbed, the length of path in the tissue concerned, and the average energy of the particles.

As a numerical example, 1 millicurie of Calcium 45 gives off 3.19×10^{12} particles per day at first (its 180-day period allows us to neglect decay for short intervals). The particles have an average energy of 85,000 volts.[8] If the calcium is taken up by the bone, all of the particles may be considered as being expended there, due to their short range (about 0.1 mm. in tissue). From this we may calculate a total energy expenditure per day of 4.3×10^5 ergs at first, or if it is homogeneously distributed, about 1000 rep per day.

Although more difficult to evaluate, the rep is a much more convenient unit for comparing radiation doses than the roentgen, because it states the exact amount of energy expended in the tissue. In the numerical example given above, the physician would emphasize the fact of equal energy doses to the two tumors by stating the dose as being about 1.1 rep/sec for each, this corresponding to 1 r in skin and 0.3 r for bone at that radiation energy. The more experimental data becomes available, the more useful will the rep prove.

The Rem. At this point another difficulty becomes apparent, in that *biological effect* is not simply proportional to the ionisation density, or energy expended. Thus 1 rep due to gamma rays may cause a homogeneous ionisation throughout the tissue, while 1 rep of alpha particles concentrates the same number of ionisations in relatively few points. The biological effect due to these identical energy expenditures may be quite different. Therefore a third unit — the roentgen-equivalent-mammal (rem)—has been introduced to allow comparison of biological effects. Thus 1 rem is the quantity of any kind of radiation producing the same biological effect as would be produced by the absorption of 1 r of X or gamma radiation [6].

As an example, 1000 r of X-rays might be observed to produce a certain biological change in a rabbit's thyroid. As in a previous example, this corresponds to about 1,100 rep. With their more localized ionisation, beta rays from iodine might produce the same effect with a dose of only 700 rep. Thus, for the beta particles in this case, 1 rem equals 0.7 rep. Were alpha particles used, their very dense ionisation might produce the same effect with a dose of only 100 rep. In this case 1 rem would equal 0.1 rep. The usefulness of the rem lies in the fact that the scientist could simply state that the effect was due to 1000 rem. Other workers, depending on their methods and equipment could translate this into 1,100 rep of gamma rays, 700 rep of beta particles, 100 rep of alpha particles, or say 600 r of a lower energy X-ray. The rem is even more difficult than the rep to assess, partly because of the difficulty of judging biological effects, and partly because of lack of experimental data for determining the rep. The results of much experimentation are now being published and the rem is coming into greater use. In particular, the dangerous idea that for all practical purposes 1 r equals 1 rep equals 1 rem is being suppressed. To determine how many r will administer 1 rem requires serious hunting through the literature, or careful experimentation.

A number of other dosage units have also been proposed, and they are based on similar considerations, and must be used with similar caution and knowledge. The *n-unit* is quantity of fast neutron radiation which produces ionisation corresponding to 1 r in the 100 r Victoreen thimble chamber.[3] In a typical case, 1 n becomes 2.5 rep in tissue, indicating the potency of neutrons The *energy-unit* (eu) is defined in a way similar to the rep, and equals about 93 ergs per gram of tissue [6] (i.e. 1:1 rep). The *J-unit* (J) defines an amount of radiation in terms of ion pairs produced, and is nearly equal to the r.[6] The *gram-roentgen* is the dose in roentgens, multiplied by the weight of the tissue irradiated.[6] It is useful for comparing whole-body doses in some cases.

In the majority of the cases to be discussed, the radiation doses were measured by observing the ionisation intensity in air, and the doses are given mainly in r. To obtain a more accurate idea of the energy dose to the various tissues, this should be translated to rep. Thus effects on the blood will be seen to be obtained with lower doses than effects on the skin, for example, but the difference is less notable when the doses are expressed in rep.

Measurement

We have seen that it is important to distinguish between the r, the rep, and the rem. The last must be evaluated by careful judgement and comparison. The rep may be calculated as outlined, and could be measured directly only with difficulty. The r may also be calculated, but in practise is more easily measured. In the case of X or gamma radiation, knowing the r usually simplifies the calculation of the rep.

The roentgen may be measured by several methods, even such cumbersome ones as that of the cloud chamber, but in practise it is almost universally measured by small ionisation chambers of the thimble type. These accumulate the charge produced in a known volume, and either indicate the accumulated charge (fountain-pen type) or indicate the rate at which it is collected (ratemeters). Since the roentgen is defined for AIR, their use implies the following assumptions:

a) That the chamber walls are made of material in which the radiation produces secondary particles of the same type and energy as in air (hence "air-wall" chambers).
b) That the walls are thick enough to produce equilibrium between the primary and secondary radiation (i.e. be several secondary mean-paths thick).
c) That the walls be thin enough to produce no effective attenuation of the photon beam.
d) That the cavity is smaller than the average range in the cavity of the ionising particles originating in the walls.

Most thimble chambers are designed so as to possess these properties over a certain useful range of photon energies. However if used for other energy ranges their readings can be quite erroneous. In using the thimble accumulating-type chambers, it is assumed that the dosage rate is constant over the period measured.

Another method of measuring accumulated dosage is to measure the exposure of photographic film carried in badges. This method is more adapted for measuring doses accumulated over long periods. It requires less expensive equipment, but very careful processing and control. [9]

Other type of measuring devices, such as geiger counters, scintillations counters, neutron counters, etc., are somewhat less versatile, but can measure a specific type of radiation very accurately. They should be used with a full appreciation of their limits. [10] For protection purposes, dosage-rate meters are to be preferred to accumulation-type instruments including film, inasmuch as these last would indicate an overdose *after* it had been received.

We see that just as the radiation unit must be well understood, the measuring instrument must be used with certain precautions:

1) The radiation measured must contain only that type which the instrument can measure, and be within the range of energies for which the instrument was designed.
2) The radiation field must be assumed constant, unless the measurement is made at the same time as the work or treatment. In the case of X-rays this is important, since the tube condition affects the intensity, and unless stabilised, the fluctuations of line voltage produce significant changes in radiation energy.

With these precautions, it is possible to measure and assess radiation dosage fairly accurately—say to 10 or 20 percent, whether in r or rep, and sometimes in rem.

There remains to be seen what dosage rate is allowable.

(Editor's Note: Conclusion of this paper will appear in the following issue (September) of the JCAR.)

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THE EFFECTS OF IODURON B ON THE RESPIRATORY TREE OF GUINEA PIGS

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Bronchial radiography, or bronchography, is an important and at times vital aid in the detection and diagnosis of various pulmonary conditions. Radio-opaque substances, such as lipiodol, presently in use for bronchography are far from ideal largely due to their quality of slow absorption.[1] This has led to a search for more satisfactory contrast substances, especially substances which are water-soluble. One of these, Ioduron B, recommended by some authors,[1] has been investigated and found to be extremely irritating to the bronchial tree.

It has not been possible to ascertain the percentage of all constituents of Ioduron B, but the following is an account of its composition. Ioduron B is a water-soluble contrast medium of high viscosity. It is a mixture of an iodine salt (Ioduron) and a thickener or viscosifier. The contrast is provided by Ioduron, which is the diethanolamine salt of diiodopyridon-N-acetic acid. The Ioduron content of Ioduron B is 50%. The thickener or viscosifier is a colloidal solution of water-soluble cellulose (sodium salt of the glycolic acid ether of cellulose). The content of viscosifier in Ioduron B is about 2.5%. Nicotinic acid amide N- acetyl bromide, which also has preservative properties, is added in order to reduce the surface tension. Since Ioduron B is vulnerable to attack by fungi and bacteria, it is necessary to add a preservative—admixture of P-oxybenzoic acid-methyl and P-oxybenzoic acid-propyl ethers. Ioduron B has a pH of 7.0–7.5 and a very high viscosity. The high viscosity (25,000 - 30,000 centipoises measured at 20° C) gives it excellent properties of adhesion and retards escape of the material from peripheral bronchioles. This largely prevents inconvenient alveolar filling.

Regarding the elimination of Ioduron B, the difference between elimination of the Ioduron content and that of the viscosifier must be recognized. Elimination of the former occurs through the kidneys, after absorption in the lung. It is not known how the viscosifier is eliminated. It is likely that a certain amount of it is absorbed directly from the lung. Certainly in most cases a considerable proportion is coughed out of the lung and expectorated[1]. Hellstrom and Holmgren[2], as a result of experiments on animals, believe that the greater part is absorbed by alveolar phagocytes and carried away. This phagocytosis is completed within one or two weeks. These two Swedish investigators, as a result of their animal experiments, do not believe that Ioduron B causes significant changes in the bronchial tree. They observed "no changes of any kind that were not completely reversible or might point to a chronically persisting irritation of the lung tissue by the contrast medium acting as a foreign body". Analagous (transitory) changes were caused by injection of physiological saline solution. Fischer on the other hand, believes that lipiodol is an injurious substance, causing irreversible changes (lipiodol granulomata).

Following the work of Fischer, five bronchograms were performed in humans at the Montreal General Hospital, using Ioduron B as contrast medium.[3] Bronchograms of good technical quality and diagnostic value were obtained. Fischer subsequently reported that he has done five hundred bronchograms in humans "without observing any damage or unpleasant side effects of any kind.[1]" He made no mention of having observed hemoptysis. In all our cases (admittedly a small series), cough was a troublesome feature during bronchography despite particularly painstaking preliminary anaesthesia. ***A more disturbing feature was the occurrence of hemoptysis in every instance.***

It was then decided to do bronchography with Ioduron B using guinea pigs as test animals. The purpose of this study was to note the effect of this contrast medium on the respiratory tree of these animals as shown by microscopic sections. The technique employed, and the results of our preliminary studies follow.

The animals were anaesthetized via the intraperitoneal route, using 0.2 c.c. of Sodium Nembutal (50 mgm. per c.c.). A cut-down was then made on the trachea, and Ioduron B injected intra-tracheally through a No. 22 needle. In some instances it proved advantageous to use a mixture of one part normal saline to four parts Ioduron B due to the smallness of the respiratory tree of guinea pigs; such a mixture flowed more easily. Entrance of the contrast material into the trachea and its distribution in the bronchi was observed fluoroscopically and radiographs were taken during fluoroscopy. In

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some cases 0.1 c.c. of 2% Pontocaine was instilled intra-tracheally prior to introduction of Ioduron B. When Pontocaine was used the animals coughed less.

In order to eliminate the possible effects of Nembutal Sodium and Pontocaine on the respiratory tree, these materials were injected separately and in combination into the bronchial tree of six animals which were subsequently sacrificed. Sections of the trachea and bronchi of these animals showed no gross or microscopic changes attributable to these substances. A "control" series of twelve bronchograms was done using Lipiodol instead of Ioduron B for the bronchography.

Bronchograms were performed on fourteen guinea pigs, with Ioduron B. Of these, five died either during bronchography or within a few minutes of the completion of the procedure. Thus, the "immediate mortality" with Ioduron B was 35.7%. The animals which died appeared to go into respiratory failure and asphyxiation, blueness of the trachea being a noticeable feature.

Bronchograms with lipiodol were performed on twelve animals. Only one animal died, and this was a respiratory death identical with that shown by the Ioduron B animals. The lipiodol "immediate mortality" was 8.3%.

Microscopic sections were made of the trachea, bronchi and lungs of the animals which died during bronchography. Other animals which survived bronchography were sacrified 5-10 minutes after the procedure and sections were made. The sections of all these animals are considered to represent the *immediate effects* of Ioduron B and lipiodol. Other survivors of bronchography were sacrificed at 24 hours to study the *delayed effects* of these substances. Still another group of survivors is being kept alive. We intend to sacrifice these animals 6 months after bronchography to study the *late effects* of the contrast media.

In this paper we are reporting the findings in the immediate group (8 with Ioduron B, 6 with lipiodol) also the 24-hour findings or delayed group (3 with Ioduron B, 3 with lipiodol).

The sections of the *Ioduron B immediate group* showed:

1. Dilatation and congestion of the vessels underlying the tracheal mucous membrane.
2. Spasm of the bronchi and sloughing of the bronchial mucous membrane.
3. Evidence of bleeding into the bronchioles and some inflammatory cell infiltration of the bronchioles.
4. Some small areas of alveolar collapse.

These changes are observed whether or not the Ioduron B has been diluted with normal saline.

The Ioduron B delayed (24 hours) group:

1. Trachea shows desquamation of the epithelial cells with cell necrosis. Hyperaemia and dilatation of the vessels beneath tracheal mucous membrane are seen.
2. Spasm of bronchioles; desquamation of bronchiolar mucous membrane.
3. Hyperaemia of bronchiolar blood vessels. Some bronchioles contain blood.
   Pyknotic nuclei seen in some of lining cells of bronchioles.

The lipiodol immediate group:

1. No tracheal reaction.
2. No sloughing of bronchial mucous membrane.
3. Some albuminoid material and a few desquamated cells found within the bronchioles.
4. Occasional red blood cell noted within the bronchioles.

The lipiodol delayed (24 hours) groups:

1. No desquamation of tracheal or bronchial mucosa.
2. Slight tracheal hyperaemia.
3. Slight bronchial spasm. Small amounts of mucous within the bronchi.

Radiographs taken at the time of fluoroscopy show that both Ioduron B and lipiodol afford adequate contrast for good bronchial visualization. Representative radiographs are included in this paper (Figs. 1 and 2). Photomicrographs of typical sections are also included (Figs. 3, 4, 5 and 6).

This is a preliminary study based on work which is still progressing. Its purpose is to report on immediate and 24-hour effects of Ioduron B on the respiratory tree of guinea pigs, using lipiodol as a control substance. Late effects will be determined in approximately six months' time. From these short term experiments, it may be concluded that Ioduron B is a damaging agent to the respiratory tree of guinea pigs. The same probably holds true for humans as hemoptysis has been observed in all patients on whom bronchography was performed, using this substance.

At present, other water-soluble contrast media are being evaluated by this method, in the hope that an entirely suitable water-soluble contrast medium for bronchography in humans will eventually be discovered. Emphasis is placed on microscopic changes produced in the bronchial tree, and the contrast qualities of the substance.

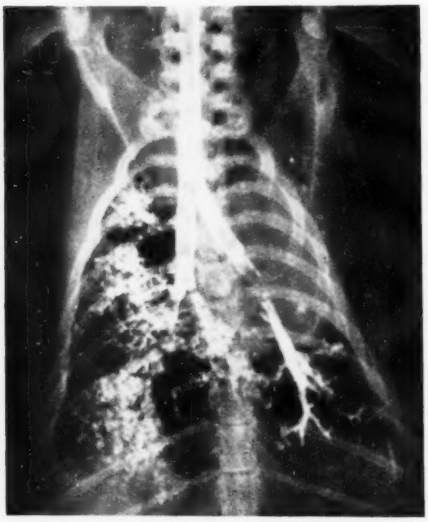

Figure 1.—Guinea pig bronchogram with lipiodol. Good contrast. Bronchial tree well demonstrated. Some alveolar filling on the right.

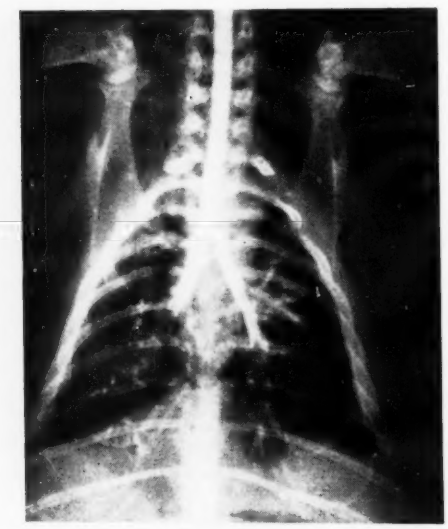

Figure 2.—Guinea pig bronchogram with Ioduron B. Good contrast, and satisfactory bronchial visualization. Film taken at an early stage of filling.

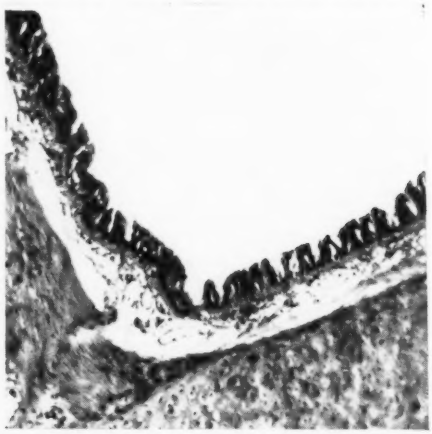

Figure 3.—Guinea pig trachea x 100, lipiodol immediate. Normal-appearing mucosa without evident reaction.

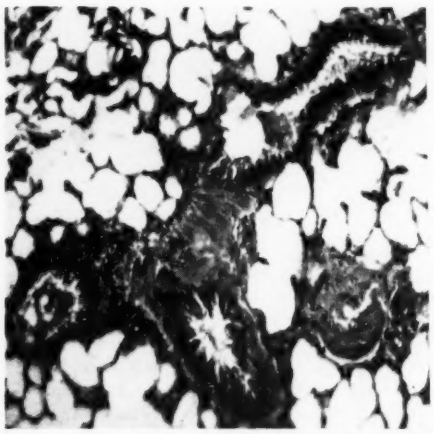

Figure 4.—Guinea pig lung x 100, lipiodol immediate. Bronchioles contain acidophilic coagulum but exhibit no appreciable reaction.

Figure 5.—Guinea pig trachea x 100, Ioduron B immediate. There is acute degeneration and shedding of mucosal epithelium, with intense hyperaemia and acute inflammatory exudative reaction in the subepithelial connective tissue.

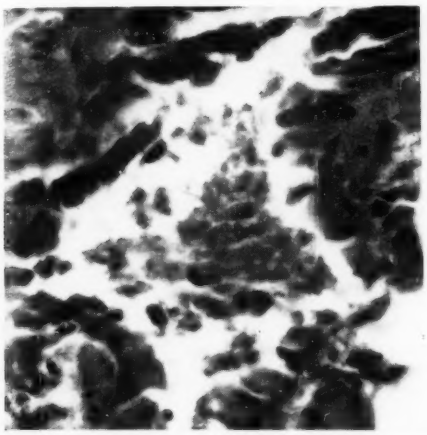

Figure 6.—Guinea pig lung x 600, Ioduron B immediate. A bronchiole exhibits epithelial degeneration and shedding with cells and granular debris in the lumen.

SUMMARY

(1) This paper is a preliminary report. The work on which it is based is still in progress.

(2) It has been shown that Ioduron B produces immediate harmful effects on the respiratory tree of guinea pigs. These harmful effects do not subside within 24 hours. Lipiodol does not produce significant changes under the same conditions.

(3) Doubt is cast on the advisability of using Ioduron B as a contrast medium for bronchography in humans.

(4) Using guinea pigs as test animals, various water-soluble opaque media are being assessed in an attempt to find one which is suitable for bronchography in humans, or to modify an existing medium in order to make it suitable for that purpose.

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RADIOLOGIST AVAILABLE

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ALVEOLAR CELL TUMOUR OF THE LUNG

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Pulmonary neoplasms have been very extensively studied and discussed during the last ten or fifteen years, and diagnostic accuracy has shown a corresponding improvement until today the diagnosis of the common forms of primary lung cancer is almost commonplace. New challenges are constantly appearing, however, especially since the widespread adoption of x-ray surveys has brought to light many lesions at a clinically and radiologically indeterminate stage. This paper is concerned with a type of primary lung neoplasm which differs very markedly in clinical and radiological features from those usually encountered. For the purposes of this report, we shall adopt the view of Paul and Ritchie[7] and of Good and others[3] that pulmonary adenomatosis and alveolar cell carcinoma are variants of the same condition. Even if this is done, it forms a very small fraction of primary lung tumors, probably somewhere between one and five per cent. Good encountered seven cases in 275 pulmonary resections for neoplasm. This lesion is of interest because of its apparent multicentric origin, and because of a remarkable morphological resemblance to a condition commonly found in sheep, known as "jaagsiekte" or Montana progressive pneumonia. The condition in sheep is undoubtedly infectious, being easily transmitted from sick to healthy animals if they are quartered together. The infectious agent has so far not been found. A similar disease has also been described in mice, horses, and guinea-pigs.

The more benign form of the disease in man is often called pulmonary adenomatosis. It is characterized by a marked proliferation of endothelial-like cells within the pulmonary alveoli, sometimes forming small papillary outgrowths into the air-cells. The process may arise in multiple areas which eventually coalesce, or may appear to spread out diffusely from a localized origin. Even the most benign-appearing form of the disease will kill the patient, since its inexorable spread eventually leaves insufficient normal lung to maintain life. Histologically however, the simplest form of the disease behaves as a benign growth. There is no invasive tendency and no metastasis to regional glands or distant areas. The disease may also be frankly malignant with local invasion and metastasis to regional lymph nodes and distant organs. There are some types between these two extremes. The largest published series of cases had metastases in about fifty per cent.

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However, there has been much confusion in the literature as indicated by the fact that all of the following terms have been used to refer to the same condition: multicentric papillary adenocarcinoma, primary multiple carcinoma, alveolar cell carcinoma, columnar cell carcinoma, alveolar cell tumour, pulmonary adenomatosis and malignant nodular carcinoma. It will be noted that these names emphasize the nodular aspect of the disease, the papillary character of the intra-alveolar growth, and the multicentric origin.

Although, as previously mentioned, there is a very similar disease in sheep, there is no published evidence at the moment to prove that the disease in man is transmissible, or that any type of microorganism is the aetiological agent.

Clinical Features

Alveolar cell tumours, like most pulmonary neoplasms, usually occur in middle or old age. It is interesting to note that the incidence is almost equal in the two sexes, (or even somewhat higher in females), in marked contrast to the situation in primary bronchogenic carcinoma which affects males at least three times as often as females. Until recently the condition was of interest chiefly to pathologists, as the diagnosis was not usually made until necropsy. At the time of Paul and Ritchie's paper in 1945[7], the diagnosis had not been made during life except in one case following lobectomy. Paul and Ritchie suggested that there were certain clinical and roentgenologic features which were sufficiently characteristic to suggest the diagnosis before death or surgical removal of the lung. The disease may begin insidiously, or more acutely with a febrile episode suggestive of pneumonia. The duration varies from a few months to several years. The most notable symptoms are cough and dyspnoea. The cough is usually productive, and in approximately thirty per cent of cases a typically profuse mucoid or watery sputum is produced in enormous amounts, up to 1500 cc. per day. This clinical finding is almost pathognomonic when present. Blood streaking or frank haemorrhage are not common except in the presence of a complicating pneumonia. Dyspnoea is *very prominent* and is frequently the first symptom. In the later stages it may become extreme. Clinical evidence of metastases is decidedly uncommon. The gradual progress of the disease causes interference with gaseous exchange in the lung, infection commonly supervenes, and

terminally there is usually a pneumonia. In the more malignant cases death may occur due to widespread metastases before gross involvement of the lungs is evident on the x-rays.

The origin of the abnormal cells is in dispute. Some believe that they arise from proliferation of a very thin epithelial layer normally present in the alveoli. Others believe that there are no alveolar epithelial cells in normal lungs and that the pathological cells arise from the epithelium of the terminal bronchioles. There appears to be good histologic evidence in support of the latter view.

Diagnostic Features

In the earlier stages, x-ray of the chest reveals a rather ill-defined area of pulmonary consolidation, suggesting a pneumonic process. The affected area is airless and more or less homogeneously opaque, although a tendency to nodularity may be evident within the shadow. Very rarely, there may be multiple discrete nodules. The condition gradually progresses, involving more and more lung until an entire lobe is involved. Still later, other foci appear in other lobes of both lungs and eventually almost all of both lung fields may be radiographically opaque. There is no atelectasis. The hilar glands usually appear normal and there is no evidence of pleural reaction. Cases with cavitation resembling abscesses do occur but are unusual. Bronchograms usually show no evidence of bronchial occlusion or bronchiectasis. A definite diagnosis is not possible from radiography alone. Bronchoscopy shows nothing definitely diagnostic, but does reveal the huge amount of bronchial secretion and permits one to obtain material for pathologic study. A trained pathologist will usually be suspicious of malignancy after examining the cells obtained, but only occasionally can make an exact histological diagnosis. The true extent of the disease at necropsy is often surprising, as abnormal alveolar cells may be found in areas which appeared radiologically normal. Evidently a good deal of the abnormal opacity seen on the film is due to abnormal secretion, and in late cases to superimposed pneumonia.

Treatment

Surgical removal of the affected lobe or lung is the best treatment available. The long term prognosis is very poor, but post-operative survivals of up to four years have been reported. There have also been reports where necropsy studies of patients dying post-operatively showed no evidence of disease in the remaining lung. Hence total removal of the disease is evidently feasible in some cases. In any case, the patient is relieved for a time of his very troublesome cough and sputum. This is worth-while palliation. No doubt some cures will be reported from time to time, as there is a growing tendency to resect any unresponsive pulmonary lesion.

Case Report

Mrs. S. C. Age 60.

Referred by private physician for x-ray of chest June 3, 1946, complaining of severe cough and sputum. She had been sick for three weeks at home, the clinical diagnosis being pneumonitis, but was feeling better.

X-ray was essentially negative. (FIG. 1.) She remained fairly well until December, 1946, when she had another episode of what was called pneumonitis and was admitted to St. Joseph's Hospital complaining of severe coughing, sputum and dyspnoea. She was in hospital a few days and was discharged. No x-ray films were taken. In June, 1947, she was again in hospital complaining of severe cough, dyspnoea, and copious sputum.

A chest film (FIG. 2) showed a hazy area of increased density in the right lower chest, obscuring the diaphragm. There was no evidence of atelectasis or hilar gland enlargement. Because it was thought that there was fluid in the right pleural space, a needle was introduced but no fluid was obtained.

In September, 1947, she was again in hospital for investigation. A bronchoscope was passed and the bronchoscopist reported as follows: "The tissues in the right main bronchus bled more easily than usual. The posterior division seemed occluded. No ulceration or definite growth could be seen. A biopsy was taken. Secretions were aspirated and sent for biopsy." Pathological report on the biopsied tissue was "suggestive of malignancy." No abnormal cells were found in the secretion.

On October 9, 1947, she was referred to Dr. E. C. Janes for surgical consultation. At that time she was coughing a great deal at night but not during the day. She brought up large quantities of *frothy* sputum. There was no blood in the sputum. She was extremely dyspnoeic or exertion. She had lost ten pounds in weight during the preceding year. She complained of pains in the upper part of the back. Physical examination revealed dullness to percussion over the lower right chest and many moist rales. The patient was sent for further x-ray examination. Preliminary P. A. and lateral chest films (FIGS. 3, 4) showed an area of homogeneous increased density occupying the area of the right middle lobe and the basilar divisions of the lower lobe. The appearance suggested pneumonic consolidation. There was no evidence of atelectasis, pleural effusion, or enlargement of hilar glands. Lipiodol was introduced and all the major and secondary bronchi were outlined and appeared normal, aside from the presence of an abnormal quantity of secretion. On November 19, 1947, the patient was operated upon by Dr. E. C. Janes. On opening the chest the right middle and

Fig. 1

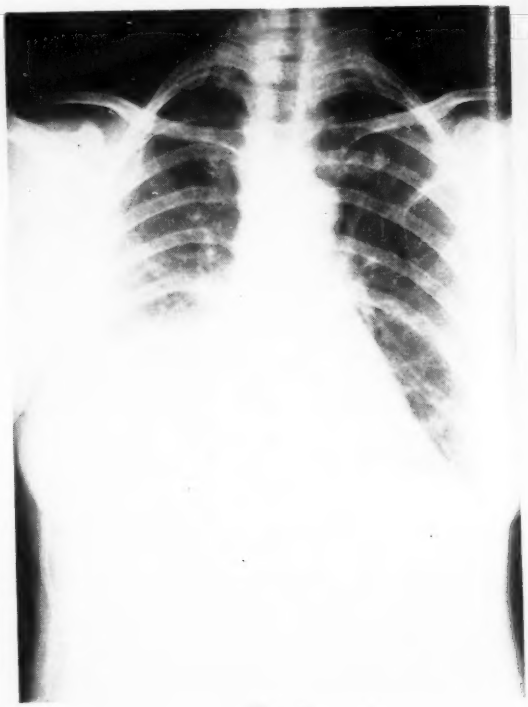

Fig. 2

Fig. 3

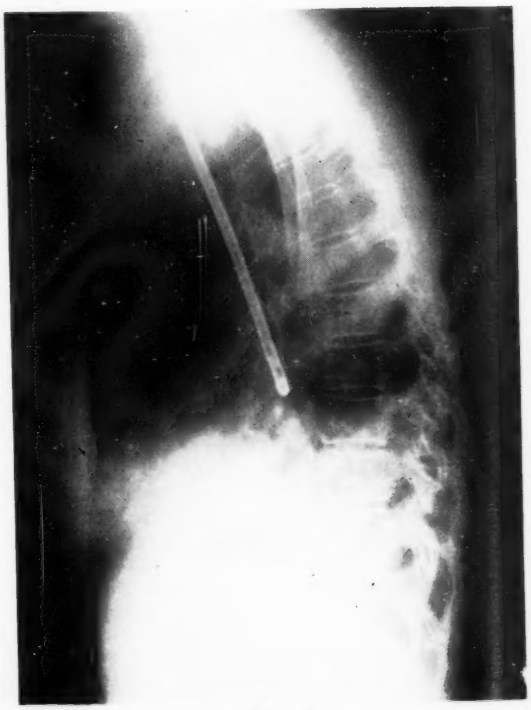

Fig. 4

Fig. 5

Fig. 6

lower lobes were dark in colour. They were very firm and looked like casts of two lobes. There was no air entry into the affected lobes. No nodules were seen. The upper lobe appeared normal. There were no pleural adhesions and no adhesions of one lobe to the other. The middle and lower lobes were resected.

Microscopic examination of the resected specimen was reported as follows: "All the alveolar walls are covered on the inside by tall columnar and cuboidal cells varying in size, shape and staining characteristics. Most of the cells are hyperchromatic but there are only very occasional mitotic figures. There are papillary projections and pseudostratification in many areas. In the interalveolar septa there is fibrosis and chronic inflammatory infiltration. The alveoli are dilated and oedema fluid is present.

Diagnosis: "alveolar carcinoma of the lung."

(This has been confirmed by several pathologists in consultation.)

This report indicates that this tumour was only mildly malignant. The patient did well postoperatively and had no complaints until May, 1948, when she again began to cough, bringing up a little frothy sputum and complaining of pain in the right chest. No definite pulmonary disease was seen on x-ray at this time.

In October, 1948, x-ray (FIG. 5) showed definite evidence of disease in the left lung, many small nodular densities being present in the lower left chest.

She was not again seen at the Clinic, but another x-ray (FIG. 6) in February, 1949, showed further progress of disease. Her attending physician stated that she went gradually downhill as both lungs gradually appeared to fill up. She died without clinical evidence of metastases in October, 1949, approximately three and one-half years from the beginning of her illness and almost two years after lobectomy. No post-mortem examination was performed on this patient.

SUMMARY

(1) A case of alveolar cell tumour of the lung treated by lobectomy is reported.

(2) The condition varies greatly in degree of malignancy.

(3) The prominent clinical features are dyspnoea, cough and copious watery or frothy sputum.

(4) The x-ray appearance suggests a pneumonic process. In any such process which does not show a normal response to treatment, if accompanied by increasing dyspnoea and the production of copious sputum, alveolar cell tumour should be suspected.

(5) Treatment by lobectomy or pneumonectomy may prolong life but cure is not to be expected.

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ANNUAL MEETING — WINNIPEG, MANITOBA

JANUARY 16th, 17th and 18th, 1952

The first mid-winter Annual Meeting of the Canadian Association of Radiologists will be held in Winnipeg on January 16th, 17th and 18th, 1952. The General Committee organizing this meeting is as follows:

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SOME OBSERVATIONS ON RENAL TUBERCULOSIS IN NEWFOUNDLAND

H. B. MURPHY, M.D.
ST. JOHN'S, NEWFOUNDLAND

Eighty-one proven cases of renal tuberculosis have been collected from those reported in the hospitals or sanitoria in Newfoundland during the three years 1947 to 1949, inclusive. Pyelographic evidence alone was not considered as proof: all have been confirmed by smear, culture, guinea-pig inoculation or surgical removal of the infected kidney. 76 of these cases came from an area of Newfoundland east of Grand Falls, (but not including it) with a population of approximately 150,000 people.

Circumstances prevent a careful statistical analysis of the morbidity from tuberculosis in Newfoundland where it is well known that the incidence of pulmonary tuberculosis is higher than in any other province of Canada. It would not be strange to find that tuberculosis of other organs or systems is frequent. Dr. Bernard Maher (St. John's) has collected 450 cases of tuberculosis of bone in the eleven-year period, 1939-1949.

Certain more chronic changes associated with tuberculosis, such as fibrosis along the urinary tract, may be frequent. The use of antibiotics (streptomycin) may accentuate this complication.

Incidence Rate

The local incidence indicated by this group of 81 cases observed in three years in markedly in excess of the relatively high-incidence series of 247 cases reported for the period 1923-1939, inclusive, by Nesbitt and others at the University of Michigan.[1] I am told by my surgical colleagues that the frequency was even higher here prior to the second world war. Dr. E. F. Moore reported in 1941 (Newfoundland Medical Convention) some 120 nephrectomies performed during 1934-1940, for tuberculosis. In the present series, 31 of 51 patients had a nephrectomy.

Fifteen of the series had unilateral renal tuberculosis, thirty-one bi-lateral disease. Two had associated nephrolithiasis, seven had calcareous deposits in the kidney.

Mortality

The majority of the lesions were far advanced. A high mortality rate might be expected, but, insofar as my follow-up has progressed, only two deaths from renal tuberculosis between 1940 and 1950 have been found. Dr. J. E. Josephson, our Pathologist, has assured me that renal tuberculosis has not been the primary cause in any death during the 3-year period under review. Some of my colleagues in Newfoundland hold to the theory that because of the high incidence of all forms of tuberculosis here, the people have acquired a relative immunity and also that the bacillus has become attenuated as to its pathogenecity. Be that as it may, the death rate (from renal tuberculosis) is extremely low and not comparable to the 50% mortality reported elsewhere.

Pathological Considerations

Tuberculosis of the kidney is commonly considered as secondary to tuberculosis in some other area of the body, usually the lungs. The classic work of Medlar[2], reviewed and checked by Thomas[3], confirms the dissemination of tubercle bacilli by the blood stream to the kidneys. Dependent upon the virulence of the organisms, the site of their deposit and the patient's resistance, the focus is either walled off or produces an active progressive lesion. "The papillary tuft is one of the first places where the tubercle bacilli produce inflammation." "Inflammatory areas may begin about the small arterioles between the tubules. If only a few bacilli are so deposited, ulceration, excavation or break-down may not ensue and no pus or bacilli find their way into the urine. The inflammatory reaction may be indicated by albumin in the urine." If the deposits are in the cortex, they may not be evident, either clinically or radiographically, for some time. Pathologically, these cortical lesions may vary from a small healed tubercle to a large tuberculoma. The latter is relatively rare. There have been only four or five reported since Bugbee's report [4] of tuberculoma in 1944. I have two cases which are believed to be tuberculoma, rather than either a cyst or a tumour.

When the focal lesion in the papilla progresses, early radiographic evidence may be forthcoming. Such constitute some 75% of clinical renal tuberculosis. The speed of extension from local excavation of the renal papilla to cavitation of the kidney substance, involvement of the individual calyces and progression into the neighbouring calyces, the renal pelvis, and thence down the ureter to the bladder, by infiltration of the submucosa or washed along by the urine, will depend upon the resistance of the host and the virulence of the organism. In a patient with relatively high resistance and

* Read before the Section of Radiology, Canadian Medical Association, Halifax, Nova Scotia, June, 1950.

low virulence of the tubercle bacilli, healing tends to occur, and the degree of subsequent fibrosis with cicatrization varies considerably. This is the type most frequent in Newfoundland.

The pyelographic evidence will include that of excavation of the renal papilla with distortion of the respective calyx, inflammation and subsequent fibrosis of the neck of the calyx, hydronephrosis of single calyces and possibly obliteration. As the disease progresses other calyces become involved, so that subsequently only a large pyonephrotic sac may remain.

Extension of the disease down the ureter and into the bladder may result in multiple strictures with intermediate stretches of ureteral dilatation or a gross general hydroureter. The hard dilated tube seen by the pathologist is the result of low ureteral stricture with subsequent dilatation and fibrosis.

Further involvement of the lower urinary tract may affect the bladder wall as the ureter passes through it, with constriction and fibrosis, or may induce any degree of cystitis from the slight inflammation about the orifice of the infected ureter to gross general inflammation with trabeculation, contraction and mucosal ulceration. This type or degree is seen in Newfoundland all too often in the advanced lesions.

Massive infection of the kidney (and urinary tract) may induce extensive calcareous deposits, or autonephrectomy.

Therapy:

Surgery

The majority of the surgeons in St. John's hold the opinion that since nephrectomy for tuberculosis in Newfoundland does not appear to carry with it the mortality reported elsewhere, and the improvement of the majority of patients so treated is so marked, unilateral nephrectomy is justified certainly in unilateral renal tuberculosis and, in bilateral disease, is preferable for the side of greater involvement, especially for removal of a demonstrable pyonephrotic sac.

Streptomycin; Chaulmoogra oil; PAS. The development of antibiotic therapy is altering the pattern of disease. Only a very few cases in the present series were treated with a combination of streptomycin and chaulmoogra oil, insufficient to reach any conclusions. It is as yet too early to formulate any judgment on the results of the use of PAS with too few patients.

The clinical improvement induced by streptomycin in our series is essentially the same as that observed elsewhere. The advanced stage of disease and degree of fibrosis in the majority prevents any marked organic improvement.

From the radiologic viewpoint, there are some ominous signs in the pyelegraphic findings. Nesbitt and Bohne [5] have called attention to the greater tendency toward cicatrization following streptomycin therapy of tuberculous lesions. Also, Lloyd[7], in a discussion of a paper by Lattimer, *et al*, [6] observed that "The healing of tuberculous lesions of the calyces and ureter may result in the formation of severe fibrous strictures which lead to a rapid destruction of the kidney under streptomycin therapy." It might be argued that hydronephrosis would have occurred even without drug therapy. But they are almost in contrast to my observations in some 100 other cases which I have examined since 1945, of whom only two showed a hydronephrosis of similar extent, and those developed only over a long period of time.

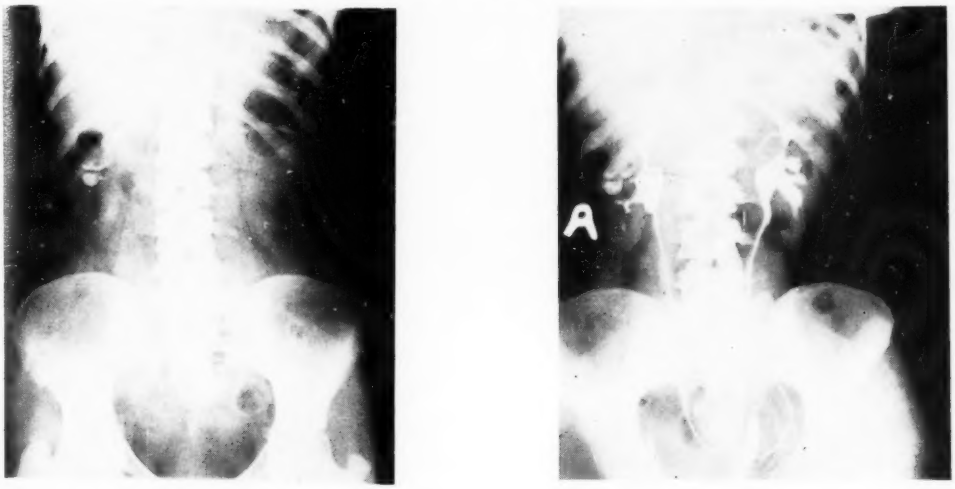
The following cases are presented briefly in illustration.

The appearance of progressive stricture of the lower ureter, or similar fibrotic distortion of the pelvis and calyces under streptomycin therapy, may perhaps be detected and its progress judged only by carefully studied periodic pyelograms.

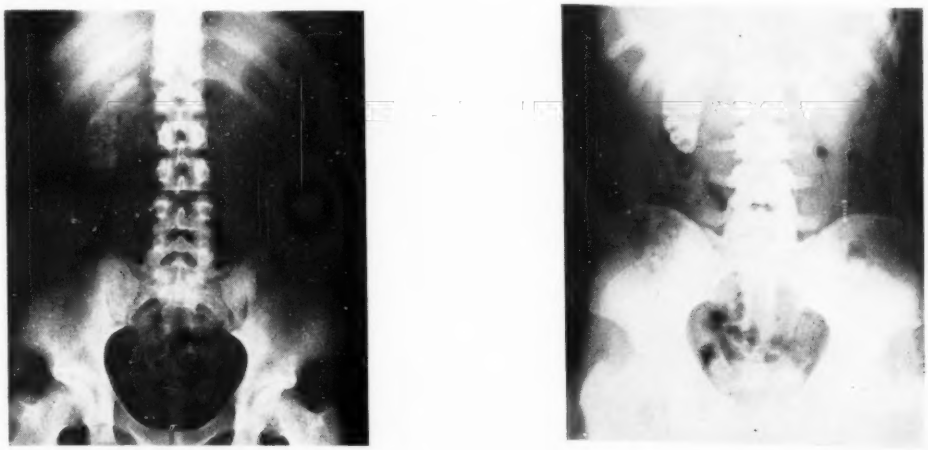
This raises the question as to whether or not we should examine routinely the urinary tract radiographically before streptomycin therapy, and regularly thereafter in order to advice the urologist or surgeon as to the effect of the treatment and or impending complications—especially in those patients from whom one kidney has already been removed.

CASE (1)

Single calyx involvement. Note clubbing of lateral inferior calyx on right. Smears positive Sept. 24/48 and Oct. 2/48. Symptoms: Frequency, urgency, ache in back.



CASE (2) Tuberculoma. Positive urine. Nephrectomy showed caseous area in cortex. Note distortion of adjacent calyces by rounded calcarious masses on right.



CASE (3) Autonephrectomy. Spine not involved. Intravenous pyelogram shows no excretion from right.

CASE (4) Cicatrization after streptomycin therapy. First pyelogram July 23/48, second pyelogram Nov. 18/49. Streptomycin started July 24/49, 1 gm. daily. Last dose Oct. 21/49. Smears negative from Aug. 29/49. Cystoscopy —*acute inflammation of bladder, moderate ulceration.*

CASE (5) Cicatrization after streptomycin therapy. First pyelogram June 13/49, second pyelogram Sept. 8/49. Streptomycin started June 23rd, ½ gm. daily, ended Oct. 26/49. Cystoscopic June 13— right orifice appeared normal. Operator was unable to find left ureteric orifice but injecting the indigo it was readily visible, a contracted *pin-hole* opening.

CASE (6) Before and after streptomycin therapy. Note decrease in hydronephrosis. Note small contracted bladder. First pyelogram Feb /48, second May/49. Cystoscopy: Capacity of bladder small.

SUMMARY

(1) As in other forms of tuberculosis in Newfoundland, the incidence of renal tuberculosis is high, but mortality from renal tuberculosis low.

(2) Nephrectomy has been employed successfully in a large number without difficulty.

(3) The pathology and associated pyelographic appearances have been discussed.

(4) There is evidence to suggest that with streptomycin therapy under certain conditions, rapid cicatrization with subsequent hydronephrosis and destruction of a kidney may occur.

(5) These suggest that more constant radiologic control studies should be made of the urinary tract in patients receiving streptomycin therapy.

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EDITORIAL

The next annual meeting of the Canadian Association of Radiologists will be held in Winnipeg during the third week of January 1952.

In the future all annual meetings will be held at the mid-winter sessions of our Association, and it is anticipated that this change will result in a greater participation by the members in the administrative activities of the Association. It means that the fiscal year will now end at the 31st of December and that the officers of the Association will be elected in January of each year instead of June. This coming annual meeting will also see the inauguration of commercial exhibits by the various manufacturers of x-ray equipment and supplies, at which an opportunity will be provided for an exchange of ideas between radiologists and manufacturers.

These are not the only important decisions which resulted from the last annual meeting of the Canadian Association of Radiologists held last June in Montreal, during the Canadian Medical Association Convention. It is understood that our Association will continue to have sectional scientific sessions and semi-annual special business meetings at the place and time of the annual meeting of the Canadian Medical Association because radiologists are physicians, and, as such, must continue to participate in the activities of the medical profession at large.

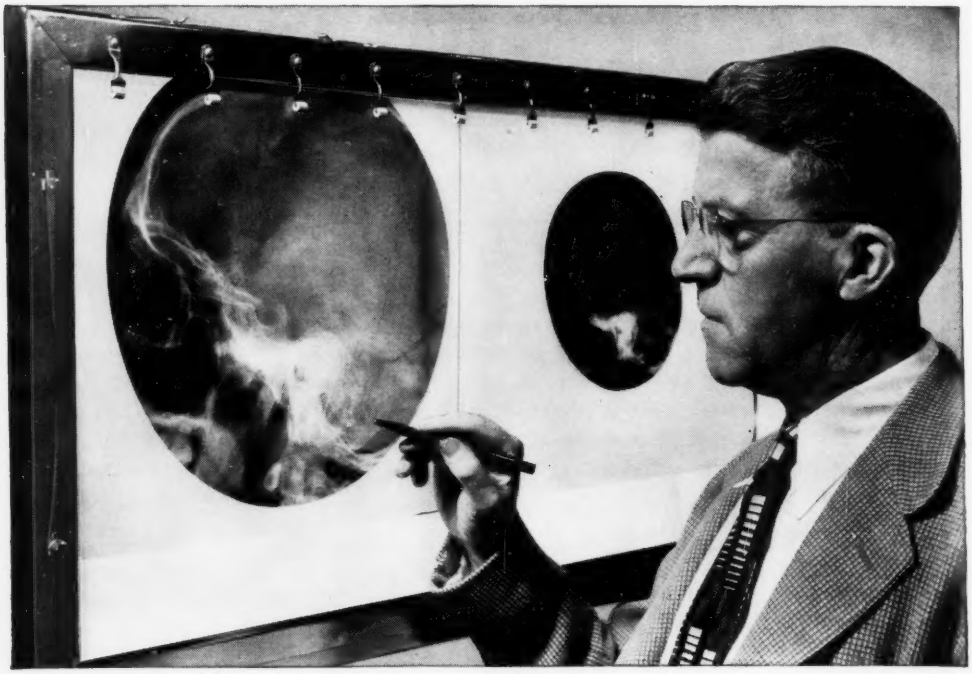
Today, with the focal point of concern on atomic warfare and defence, we are endeavouring to meet our responsibility to the people of Canada, as a scientific body, through the formation of a Committee on Atomic Defence. This Committee will consist of two radiologists and one physicist, and will offer the services and co-operation of the Association to the Federal Government in the organization and regulation of defence against atomic weapons.

The Winnipeg Meeting will coincide with the 15th anniversary of our Association. It was in 1937 that the Canadian Association of Radiologists was organized from an informal meeting held under the sponsorship of the late Dr. G. E. Richards.

An anniversary always brings an excellent opportunity to look back and review what has been accomplished. Our original membership has more than doubled; our Association has grown into thirteen active divisions across Canada, and is now governed by a federal charter. Certification and fellowship in Radiology and its branches have developed in the Royal College of Physicians and Surgeons of Canada. Post-graduate training in Radiology has become organized in Canada. Progress has also been made in the gradual strengthening of inter-specialty and hospital-radiologist relations, resulting in better understanding of mutual problems. The scientific contributions of Canadian radiologists have justified the publication of the *Journal of the Canadian Association of Radiologists*, since 1950.

An anniversary brings also an opportunity to look forward to the future. There is, of course, still much to be done, and while we acknowledge this fact, let us keep it foremost in our minds by remembering John Ruskin's words:

"When we build, let us think that we are building forever. Let it not be for present delight nor for present use alone. Let it be such work as our descendants will thank us for, and let us think, as we lay stone on stone, that a time is to come when those stones will be held sacred because our hands have touched them, and that men will say as they look upon the labor and wrought substance of them 'See! This our Fathers did for us.'"



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